

Modern Concepts of Cardiovascular Disease

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ROENTGEN RAY INTERPRETATION OF CARDIOVASCULAR DISEASE

Part I

The examination of the cardiovascular system by means of the roentgen rays is but one amongst several methods of study of the circulation. To disregard this method means partially to neglect our best trained sense, the eye. Roentgen ray examination has rightly been called *autopsia in vivo*. Its value as well as its limitation must be evaluated by basic knowledge of the normal and pathological anatomy and physiology of the cardiovascular system on the one side, and of the fundamental, theoretical and applied physical principles of the roentgen rays on the other. Deficient training and deficient insight into problems will only lead to disappointment and to misinterpretation of results as well as of possibilities.

Every roentgen ray study has to start with fluoroscopy. Rotating the patient under fluoroscopy has the same relation to a film record as a ride through the country has to a picture post-card of the same scenery. A film record in its frozen aspect expresses the statics, while fluoroscopy expresses the dynamics of the situation. Unfortunately many workers do not adhere to the fundamental rule of waiting in a completely darkened room until their eyes are fully adapted; on the average at least fifteen minutes are needed. If only fluoroscopy is wanted, the smallest and cheapest apparatus will do; self-protective and water-cooled X-ray tubes are needed. A well exposed film taken at seven feet distance in the usual anterior projection, or at four feet distance in an oblique position should not require more exposure than 1/15 second, which means that high powered equipment is necessary. Orthodiagraphy consists in taking a tracing of the shadow of the heart and great vessels outlined against the fluoroscopic screen by the central rays from the roentgen tube. A copy on transparent paper is kept for permanent record. Only those should use this method who have received training in it and can practice it constantly. Fluoroscopy or film records in the postero-anterior position only are insufficient. Films are taken at the angle of rotation of the patient which gives most information. Study during deep respiration, in lateral positions of the body and study of the barium filled

esophagus give additional information.

The chief difficulty lies in recognizing borderline cases. Therefore, at first, experience as to what is "normal" has to be gained. The "normal" is a logical rather than a biological concept and should be replaced by the term "range of normal variation." Anatomically one may predict, for instance, that the circumference of the aorta for a person at the age of 20-25 and the height of 1.75-1.85 meters should be 5.7 cm., yet it has been shown during the world war in postmortem studies on previously healthy soldiers that the normal aorta may measure from 5.1 cm. to 7 cm.

Absolute figures have little but statistical value. It is advisable to correlate them with other figures; for instance, in a given person, a certain diameter of the heart silhouette may be related to body weight and chest circumference. A fallacy of heart measurement in the anterior plane is that the antero-posterior (sternovertebral) dimension of the heart is quite at variance as compared with any diameter taken in the frontal plane. The transverse diameter of two hearts may be 13 cm., yet the depth of the one heart may be 8 cm. and of the other one 10.5 cm. Thus their volumes are quite different.

Though a certain amount of information can be gained in observing the pulsating heart, it must be remembered that the mechanics of the heart would be poorly understood if the movement of the atrioventricular septum were not considered. This is the plane in which the heart valves are located. Here is a real "pump" at work, moving toward the apical portion during systole. This cannot be seen normally, but is well visualized when calcareous deposits are present, as sometime occur, for instance, along the mitral ring. It follows from this conception that an attempt to determine the cardiac output of the heart by comparing the systolic and diastolic size of the heart silhouette is unreliable.

The roentgen picture of the central cardiovascular system always must be correlated with the constitutional type of the individual, just as is the case with the roentgenological study of the gastro-

intestinal tract. In people with the symptom complex of neurocirculatory asthenia we see a rather small, vertical, rapidly contracting heart. If the circulation is improved due to change into the recumbent position, or by increasing the abdominal pressure, then we see an increase in the size of the cardiac silhouette together with a slowing of the pulse rate, the hydromechanic conditions having been improved.

It is obvious that in obesity, emphysema and in chest deformities, the results of percussion become most unsatisfactory. But also for percussion in general, the objective check-up by the roentgenological method—in drawing the orthodiagraphic silhouette directly on the skin of the patient—is of great teaching value. One can state that with both methods similar results are obtained as to the outline of the heart (normal, mitral, aortic). The right heart border as revealed by percussion is always found to be inside of the roentgenological heart border; the explanation for this fact is that the right atrium is relatively thin walled and relatively distant from the anterior chest wall. The clinical apex beat may coincide with the roentgenological apex of the heart. If this is not the case, the former one will be found more often to be outside than inside of the latter one. The determining factors for these differences are the degree of the curvature of the chest and the radiation of the palpable apex beat which is quite marked in the hyperactive heart. A comparison between the roentgenological findings and clinical percussion results shows that under normal conditions the great vessels—aorta, pulmonary artery and superior vena cava—cannot be reliably percussed. It is the experience of the author that the great majority of physicians, by their percussion, find the heart and great vessels too high up in the chest.

Roentgen ray studies are applied with increasing frequency to those who are engaged in athletics. It is a moot question whether strenuous physical exercise may produce cardiac dilatation or hypertrophy. It may be that only those hearts are affected which have been damaged previously, for instance by infectious diseases. It is possible, furthermore, that hearts which are far off in the distribution curve of the normal figures as to their size, hearts which are at the limits of the normal to start with, are responding in the above mentioned sense. The fact is that in certain types of athletics (rowing, bicycling, skiing) a small percentage actually will demonstrate a heart size exceeding the upper limit of the normal. In these cases, serial examinations by roentgen rays at intervals of about four weeks are of utmost importance. Changes, for instance, of 1 cm., either an increase during training or a decrease after having stopped training, cannot be registered by any other method. These films must be taken and repeated under standard conditions, otherwise any deductions and conclusions are valueless.

One way of presenting our topic would be to

elicit the roentgen ray findings, positive or negative, along with an enumeration of the causes and types of heart disease, structural abnormalities and disorders of function. The other way, which has been chosen here, is to start out from the viewpoints of size, shape, position, and pulsatory movements of heart and great vessels; in addition the relation to and the appearance of adjacent organs will be discussed.

To start with, certain pathological changes or functional states give negative roentgen ray findings. As an example of a disease with negative X-ray findings, we may cite coronary disease with angina pectoris but without myomalacia and without cardiac enlargement; as another, uncomplicated interventricular septal defect; as a third, an early state of endocardial valvular lesion; as a fourth, early luetic aortitis.

Cardiac enlargement which affects both ventricles or all heart chambers equally may be caused by a number of underlying conditions. The following are examples: changes in the myocardium due to hypertensive cardiovascular disease with an accompanying enlargement of the right side; endocrine disturbances like myxoedema, thyrotoxicosis, hyperpituitarism; infectious-toxic states like diphtheria; pernicious anemia; avitaminosis like beriberi; a metabolic disturbance in the form of an abnormal glycogen storage; a traumatic or congenital arteriovenous anastomosis; combined valvular lesions following rheumatic heart disease. Yet a detailed analysis will enable us to draw distinctions and to separate them into different divisions. Thus the pulsations in anemia and thyrotoxicosis are alike but entirely different from those seen in the myxoedema heart or in the failing myocardium. The combined valvular lesion will show an increased amount of blood in the lungs, whereas this is not the case in the presence of thyrotoxicosis or beriberi, even in the presence of cardiac decompensation. Often instead of a general enlargement of the heart, certain anatomical parts of the heart will be enlarged. Here it may be mentioned that some characteristics in the silhouette exist which enable us to differentiate between earlier and later stages of right and left ventricular enlargement respectively. If a ventricle has to work continuously against abnormal resistance, first the so-called outflow tract, i.e., only the part from the apical portion toward the respective semilunar valves will be affected, mainly in the form of increase in length of the ventricle. Whereas in a later stage, the inflow tract, i.e., the part from the apical portion towards the respective atrioventricular valves will participate also, the result being a more globular enlargement of the respective ventricle.

(To be continued)

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